Title: Affective and Autonomic Reactivity During Parent-Child Interactions in Depressed and Non-Depressed Mothers and Their Adolescent Offspring

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Abstract

Objective: Determine how depressed mothers and their adolescent offspring differ in affective and autonomic reactivity during positive and negative parent-adolescent interactions.

Methods: In this multi-method case-control study of 180 mother-adolescent dyads (50% mothers depressed, 50% mothers non-depressed) we collected self-reported positive and negative affect, as well as measures of cardiovascular and electrodermal autonomic activity during two mother-adolescent interaction tasks designed to differentially elicit positive (Event-Planning Interaction; EPI) and negative (Problem-Solving Interaction; PSI) affect.

Results: Findings indicated that both depressed mothers and their adolescent offspring exhibited greater self-reported negative affect reactivity during the negative interaction and blunted sympathetic activity as measured via skin conductance level across both positive and negative interactions. These effects persisted after controlling for a range of potential covariates, including medication use, sex, age, adolescents own mental health symptoms, and behavior of the other interactant and results remained after correcting for multiple comparisons.

Conclusion: These findings indicate that depressed mothers and their adolescent offspring exhibit similar patterning during parent-child interactions, including increased negative affect reactivity during negative interactions and blunted sympathetic activity across both positive and negative interactions. These findings have potential implications for understanding the role of family processes in the intergenerational transmission of risk for depressive disorders.

Introduction

Depression is associated with significant impairment, morbidity, and mortality, which presents risks that are profound and intergenerational. When those who are affected are parents, it adversely affects family interactional processes and relationships, creates transdiagnostic psychiatric risk amongst offspring, and has adverse physical health outcomes for both the depressed individuals and their offspring (Nelson et al., Preprint; Nemeroff & Goldschmidt-Clermont, 2012; Raposa et al., 2014). Importantly, research on the association of depression with physiological processes that might be associated with physical health, however, is rarely contextualized within the family environment, limiting our understanding of how risk is embedded in fundamental daily processes such as family interactions. In this study, we examined the influence of maternal depression on affective and autonomic reactivity amongst mothers and their adolescent offspring—variables with significance for both mental and physical health—during parent-adolescent interactions.

Association of Depression with and Affective and Autonomic Stress Reactivity

Affective Reactivity. Depression has clearly been found to be associated with higher levels of baseline negative affect and lower levels of baseline positive affect (Davidson et al., 2002; Forbes et al., 2004; Watson, 2000), but the story becomes more complicated when considering affective reactivity. In a systematic quantitative meta-analysis, Bylsma and colleagues (2008) discuss three competing hypotheses regarding the influence of depression on affect reactivity, including: 1) reduced positive reactivity (i.e., positive attenuation); 2) increased negative reactivity (i.e., negative potentiation); and 3) emotion context insensitivity (ECI; i.e., positive and negative attenuation; Rottenberg, 2007; Rottenberg et al., 2005), and conclude that laboratory studies provide most support for ECI. One issue in this research, however, is that

most studies included in the meta-analysis used stimuli of low personal relevance, such as pleasant or unpleasant pictures or film clips. Another body of literature, demonstrates that those with depression exhibit greater reactivity to emotional stimuli that is social in nature and indicative of threat (N. B. Allen & Badcock, 2003; Price et al., 1994). Similarly, other researchers have postulated that this ECI profile is limited to lab-based research (Nelson, Klumpp, Doebler, & Ehring, 2018; Thompson et al., 2012) and may not be present in real-world or ecologically valid contexts, particularly in regards to negative affect. More particularly, research shows that those with depression have greater NA reactivity to negative life circumstances, such as during unpleasant events (Bylsma et al., 2011; van der Stouwe et al., 2019); daily life disturbances (Myin-Germeys et al., 2003); subjective stress (Bylsma et al., 2011); and daily event, activity, and social stress (van Winkel et al., 2015). In non-clinical samples increased NA reactivity during daily life stress has been found to be associated with higher vulnerability for depression (Wichers et al., 2007). Furthermore and in line with this reasoning, stress sensitivity or the tendency to react stronger to stress has been shown to predict increased depressive symptomatology and development of depression (Wichers et al., 2007, 2009), from which we would infer that those with depression would have greater NA reactivity. Moreover, research has shown that those with depression have comparable levels of PA reactivity compared to non-depressed individuals (Heininga et al., 2019; Thompson et al., 2012), although these findings are not found in all studies (Peeters et al., 2003).

Overall, remaining questions include how both PA and NA reactivity are altered in those with depression based on research design (lab versus real world), type of event (positive or negative), type of stimuli (interpersonal or not personal), and emotional valence of the

interpersonal interaction (positive or negative). More research is needed to tease apart these contextual and stimuli effects.

Autonomic Reactivity. Depression is also associated with psychophysiological stress reactivity (Bylsma et al., 2008, 2014; Koval & Kuppens, 2012; Koval et al., 2012; Nelson, Byrne, Sheeber, et al., 2017), with most research finding blunted autonomic responses as depressive symptoms increase (Carroll et al., 2007; Phillips, 2011; Salomon et al., 2009; Schwerdtfeger & Rosenkaimer, 2011; Stange et al., 2017; York et al., 2007), although some findings have been mixed, with regard to the direction of effects (Kibler & Ma, 2004; Light et al., 1998; Matthews et al., 2005; Stange et al., 2017). As Schwerdtfeger and Rosenkaimer (2011) point out, these mixed findings may be due to type of stressor (active vs. passive). Active stressors, which include interpersonal interactions (e.g., public speaking tasks), result in a beta-adrenergic response that yield blunted autonomic reactivity (Carroll et al., 2007; Phillips, 2011; Salomon et al., 2009; York et al., 2007). Such a blunted pattern of biological disengagement has been confirmed in a recent review of depression and autonomic reactivity (Ginty, 2013; Schiweck et al., 2019), specifically for sympathetic activity (Sarchiapone et al., 2018), and is in line with motivational deficit in those with depression (Kristen Salomon, Bylsma, White, Panaite, & Rottenberg, 2013; Schwerdtfeger and Rosenkaimer, 2011). Furthermore, as Schwerdtfeger and Rosenkaimer (2011) point out, passive stressors (e.g., mirror tracing task), on the other hand, engender an alpha-adrenergic response that results in increased autonomic reactivity (Matthews et al., 2005). Overall, more research is needed to replicate these patterns of blunted autonomic reactivity during active stressors across the autonomic systems in order to identify reactivity patterning during important interpersonal interactions that are both positively

and negatively charged within the family environment to increase the limited understanding of how risk is embedded in daily processes of family interactions.

Maternal Depression as a Potential Risk Factor for Offspring Affect and Autonomic Reactivity

As mentioned above, depression not only presents profound risks for the individual, but also intergenerational risks, with adverse health outcomes for both depressed persons and their offspring. There are various potential pathways through which maternal depression may act as a risk factor for offspring affective and autonomic reactivity. These include both genetic mechanisms, which may account for up to 40% of the variance in depression (Goldberg, 2006; Levinson, 2006), as well as adverse environmental factors, including exposure to higher rates of negative parenting behaviors displayed by depressed mothers (Beck, 1995; Leinonen, Solantaus, & Punamaki, 2003; Norcross, Leerkes, & Zhou, 2017). These negative parenting behaviors include greater hostility, irritability, negative affect, intrusiveness, criticism, unpredictable behavior, withdrawal and unresponsiveness (Leinonen, Solantaus, & Punamaki, 2003; Norcross et al., 2017) -- all of which are likely to be experienced as stressful by offspring, potentially leading to affective and autonomic dysregulation. Nonetheless, to date few studies have examined the association between maternal depression and affective or autonomic stress reactivity in offspring (Bleker et al., 2018). In the few available studies, findings suggest that prenatal and early postnatal exposure to maternal depression is associated with greater autonomic (e.g., systolic blood pressure) reactivity in middle childhood (Fan et al., 2016), and adolescence (Vedhara et al., 2012), although consistent findings are not observed in all studies (Rash et al., 2015). Furthermore, some recent research has shown that exposure to prenatal maternal depression is associated with blunted child sympathetic (i.e., electrodermal) activity

(Buthmann et al., 2019). Other research has found that prenatal depression is associated with greater infant negative behavioral activity in response to novel stimuli (Davis et al., 2004). Research on offspring exposed later in life has demonstrated mixed findings with some studies showing greater physiological reactivity to stress (Burkhouse et al., 2014; Gump et al., 2009) and some not (Waugh, et al., 2012), though the latter study did show greater NA reactivity (Waugh et al., 2012). The broad range of ages, stimuli, and outcome measures, in conjunction with the relatively small evidence base, moreover, makes it difficult to draw clear conclusions.

The Current Study

The current study examined cross-sectional differences in affective and autonomic reactivity in mothers with and without depression (Depressed and Non-Depressed Group) and their adolescent offspring (High Risk Group for those with mothers experiencing depression and Lower-Risk Group for those with mothers not experiencing depression). We examined reactivity during two mother-adolescent interaction tasks designed to differentially elicit positive (Event-Planning Interaction; EPI) and negative (Problem-Solving Interaction; PSI) interpersonal processes, and which we have found to elicit differential autonomic responses in past research (Nelson et al, 2017). We examined tasks that elicit both PA and NA, given that both positive and negative contexts may be stressful to depressed persons (Forbes et al., 2004; Watson, 2000), and that our prior work has revealed that harsh parental behavior during putatively positive affective contexts have adverse associations with adolescent emotional health (Schwartz et al., 2013).

Affect Hypotheses. We hypothesized that during both positive and negative interactions, Depressed mothers and their High-Risk offspring would report blunted PA reactivity compared to Non-Depressed and Lower-Risk Groups in accordance ECI theory and past research. We hypothesized that, across interactions, Depressed mothers would show greater NA reactivity

compared to Non-Depressed mothers based on research indicating greater stress sensitivity and reactivity to social stimuli. In addition, we hypothesized that during positive and negative interactions, High-Risk offspring would show greater NA reactivity as maternal depression may act as a form of social threat for adolescents that results in increased in affective reactivity during mother child interactions.

Autonomic Hypotheses. We hypothesized that across interactions, Depressed mothers would be less reactive -- as indicated by lower heart rate reactivity, less shortening of pre-ejection period, greater heart rate variability, and lower skin conductance level compared to Non-Depressed and Lower-Risk Groups as prior research has shown that active stressors result in blunted autonomic activity in those with depression. We do not have specific hypotheses to how this may differ by type of interaction task and therefore, this is an exploratory analysis.

In contrast, we hypothesized that across interactions, adolescents of depressed mothers (High-Risk Group) would have greater heart rate reactivity, more shortening of pre-ejection period (i.e., shortening of interval), less heart-rate variability, and greater skin-conductance reactivity relative to adolescents of non-depressed mothers (Lower-Risk Group) as our adolescent hypotheses are based on the premise that maternal depression acts as a form of social threat for adolescents that results in increased physiological reactivity during mother child interactions.

Methods and Materials

Participants

Participants were 180 low-income women (see Table 1) and their adolescent children, aged 11-14 (see Table 2). Two groups of women were recruited: a Depressed Group, selected for currently elevated depressive symptoms and a history of treatment for depression and a Non-

Depressed Group, selected for no or low levels of current depressive symptomatology, no history of treatment for depression, and no current (i.e., past month) mental health treatment for any mental health disorder. Their adolescent offspring were considered at High- and Lower-Risk, respectively for disturbances in affective and autonomic reactivity. Exclusion criteria for mothers and adolescents in both groups included psychosis, cognitive impairment, or other conditions that would interfere with meaningful participation. There were no statistically significant group differences in adolescent biological sex, adolescent age, race, and ethnicity or maternal education, employment, or income.

Table 1. Maternal Characteristics by Group

Variable	Depressed			Non-Depressed			Group Differenc e
	<i>N</i>	Mean (<i>SD</i>)	Percentage	<i>N</i>	Mean (<i>SD</i>)	Percentage	<i>p</i> -value
Group	90		100%	90		100%	
Age	90	40.6 (6.5)		90	40.0 (6.4)		.482
Mental Health							
Depressive	90	12.32		89	2.57		< .001
Symptoms		(5.84)			(2.70)		
Anxiety	90	9.69		90	2.34		< .001
Symptoms		(5.42)			(2.87)		
Employment							.077

Currently employed	44	48.89%	57	63.33%
Homemaker	23	25.56%	21	23.33%
Disabled and unable to work	9	10.00%	1	1.11%
	8	8.89%	8	8.89%
Underemployed, looking for a job				
Currently a student	3	3.33%	1	1.11%
Other	3	3.33%	2	2.22%

Education Level .073

Less than High School	3	3.33%	3	3.33%
High School Graduate/	8	8.89%	17	18.89%
GED				
Vocational or Professional School	7	7.78%	8	8.89%

Certificate/					
Some College	57	63.33%	39	43.33%	
Bachelor's	15	16.67%	23	25.56%	
Degree or					
Higher Degree					
Income					.484
<\$17,000	25	27.78%	16	18.18%	
\$17,000 -	7	7.78%	13	14.77%	
\$19,999					
\$20,000 -	14	15.56%	8	9.09%	
\$24,999					
\$25,000 -	11	12.22%	10	11.36%	
\$29,999					
\$30,000 -	5	5.56%	6	6.82%	
\$34,999					
\$35,000 -	6	6.67%	9	10.23%	
\$39,999					
\$40,000 -	8	8.89%	10	11.36%	
\$49,999					
>= \$50,000	14	15.56%	16	18.18%	
Don't Know	0	0.00%	2	2.22%	

Table 2. Adolescent Characteristics by Group

	High Risk			Low Risk			Group
Variable	<i>N</i>	Mean (<i>SD</i>)	Percentage	<i>N</i>	Mean (<i>SD</i>)	Percentage	Difference <i>p</i> -value
Group	90		100%	90		100%	
Age	90	12.90 (1.30)		90	12.90 (1.20)		.455
Adolescent Sex							.968
Male	45		50%	51		56.67%	
Female	45		50%	39		43.33%	
Adolescent Gender							
Male	45		50.00%	52		57.78%	
Female	44		48.89%	36		40.00%	
Other	1		1.11%	2		2.22%	
Race							.294
White or Caucasian	75		83.33%	67		74.44%	
More than One Race	15		16.67%	18		20.00%	

American Indian/ Alaska Native	0	0.00%	1	1.11%
Native Hawaiian/ Pacific Islander	0	0.00%	1	1.12%
African American	0	0.00%	1	1.11%
No Response/ Unknown	0	0.00%	2	2.22%
Ethnicity				.248
Not Hispanic or Latina	77	85.56%	70	77.78%
Hispanic or Latina	13	14.44%	20	22.22%

Recruitment

In order to recruit a low-income sample, the majority of participants ($n = 132$) were recruited through the organization that administers the Oregon Health Plan (Medicaid) in the county where data were collected. The remainder of the sample ($n = 48$) were recruited through online advertisements; those recruited online were screened to ensure that their incomes would

have rendered them eligible for Medicaid. This low-income sample was selected, because mental health problems such as depression are more prevalent in these groups. There were significant group differences in recruitment source, such that more mothers in the depressed group were recruited online, $\chi^2(1) = 12.528, p < .001$. Mothers and adolescents provided informed consent and assent, respectively, prior to assessment. All procedures were approved by the Institutional Review Board.

Assessment Procedures

After an informed-consent meeting, mothers completed an online questionnaire through a secure link and diagnostic interviews were conducted over the phone by research assistants. Subsequently, mothers and adolescents participated in a laboratory assessment where participants completed questionnaires and were outfitted with ambulatory electrocardiography (ECG) and impedance cardiography (ICG) devices to record psychophysiological indices during a 2-minute baseline and then participated in two 15-minute interaction tasks. One task was an Event-Planning Interaction (EPI) in which they were asked to plan a vacation they would like to take together. The second task was a Problem-Solving Interaction (PSI), in which dyads were asked to discuss and try to resolve one or two areas of conflict selected from the Issues Checklist (Prinz et al., 1979). Topics chosen for discussion were those with the highest mean frequency by intensity ratings across mother and adolescent reports. These tasks have been shown to differentially elicit positive and negative affect, respectively (Nelson, et al., 2017). The ordering of tasks was counterbalanced, and separated by a puzzle task to reduce affect contagion or carry-over effects from one task to the next.

Measures

Diagnostic Measure.

Mothers completed the Structured Clinical Interview, non-patient version (SCID-NP; First, Spitzer, Gibbon, & Williams, 1996) to ensure that participants in the non-depressed group did not meet criteria for current or past depressive disorder. Overall interrater reliability for this study was $\kappa = .80$.

Self-Report of Mental Health Symptoms.

Mothers completed the Patient Health Questionnaire-9 (Kroenke, Spitzer, & Williams, 2001) and the Generalized Anxiety Disorder-7 (Spitzer et al., 2006).

Affect Assessment.

At the start and conclusion of each interaction, mothers completed the Positive and Negative Affect Schedule (Watson et al., 1988) and adolescents completed the Positive and Negative Affect Schedule for Children (PANAS-C; Laurent et al., 1999), respectively, to provide indices of positive affect (i.e., energy, interest, and engagement) and negative affect (i.e., emotional distress) in order to measure affective reactivity during interaction tasks.

Behavior Assessment.

The Living in Family Environments (LIFE) coding system (Hops, Biglan, Tolman, Arthur, Longoria, 1995) was used to code mother and adolescent behavior during the video-recorded dyadic interactions. Three constructs -- aggressive, dysphoric, and positive interpersonal behaviors were created based on verbal content and nonverbal and paraverbal affect. Variables created reflected the proportion of the interaction that each person engaged in the behavior during the task. Observers were blind to group. A second observer coded 20% of interactions to provide estimates of interobserver consistency. The average interclass correlation

(ICC) for maternal behaviors was 0.90 with a range from 0.72 (aggressive behavior during EPI) to 0.97 (dysphoric behavior during PSI) and the average ICC for adolescent behaviors was 0.88 with a range from 0.82 (aggressive behavior during PSI) to 0.93 (dysphoric behavior during EPI). Further details on the LIFE coding system can be found in Supplementary Materials.

Biological Measures

Psychophysiological Assessment. ECG and ICG data were acquired using Vrije Universiteit Ambulatory Monitoring System, which uses a 3-lead ECG and 4-lead ICG. Data were scored using the Data Analysis and Management Software (DAMS) program (<http://www.vu-ams.nl/>). To obtain the resting baseline assessment, participants were asked to sit quietly for a 2-minute period. Data were averaged over this 2-minute period.

Autonomic Activity: Heart Rate was calculated based on time (in milliseconds) between successive R waves (R-R intervals) on the ECG to calculate average beats per minute. The root mean square of successive R-R interval differences (RMSSD), which has been shown to reflect vagal tone (Kleiger et al., 2005; Laborde et al., 2017; Thayer & Lane, 2007) was calculated to measure parasympathetic nervous system activity. Cardiac pre-ejection period (PEP), a marker of sympathetic nervous system (SNS) activity, was automatically calculated for each cardiac cycle as the time interval in msec between the onset of ventricular depolarization (Q wave onset of the ECG) and the opening of the aortic valves (B point in the ICG dZ/dt signal). Skin conductance level (SCL), a second measure of SNS activity, was automatically calculated based on the electrodermal activity of the skin, which was measured using direct current (DC) utilizing a 16 bit A/D converter.

Covariates. We collected the following measures as potential covariates: age, self-reported biological sex, ethnicity, race, body mass index, and medication use. Only covariates

that were significantly associated with outcome variables were included in analyses in order to prevent overfitting.

Statistical Analyses.

All statistical analyses were conducted with R Studio, version 1.1.463. See supplemental materials for statistical code and packages used for analyses. Statistical significance was defined using 95% confidence intervals and *p*-values. Exploratory analyses including histograms as well as skew and kurtosis statistics were run for each variable to check for normality. Any variable that had a skew of +/- 2 was log transformed. All variables were winsorized to +/- 3 SD to correct for outliers. All tables for unadjusted and adjusted models as well as figures for null results can be found in supplementary material.

To assess differences in reactivity a series of MLM models were performed for each cardiovascular measure (HR, RMSSD, PEP, SCL) and affect measure (positive and negative) separately for adolescents (High-Risk and Lower-Risk Groups) and mothers (Depressed and Non-Depressed Groups). For each model, intercepts were allowed to vary by participant. For each of the adolescent analyses, three separate models were run for each outcome adding more stringent covariates, which included 1) unadjusted model with only baseline physiology or affect, group, task, and group x task as fixed effects, 2) adjusted model with age, biological sex, and medication entered as fixed effects, and 3) adjusted model with adolescent's own mental health symptoms added as a fixed effect in addition to prior covariates in order to covary out any influence adolescents' own mental health had on outcome measures. For each of the mother analyses, two separate models were run, and for each more stringent covariates were successively added, which included 1) unadjusted model with only baseline physiology or affect, group, task, and group x task as fixed effects and 2) adjusted model with age and medication

entered as fixed effects in addition to prior covariates. Dependent variables included measures of affect (positive and negative) and psychophysiology (heart rate, RMSSD, PEP, and SCL) averaged within each condition. Significant interactions were explored descriptively using graphical presentation of the data and post-hoc statistical tests of differences between conditions with simple contrasts. All reactivity model tables can be found in supplementary materials.

We corrected for multiple comparisons by hypothesis construct, such that we ran Benjamini-Hochberg Correction (Benjamini & Hochberg, 1995; Benjamini & Yekutieli, 2001) for pre-ejection period and skin conductance level analyses (sympathetic nervous system hypotheses). We did not correct for multiple comparisons for PA and NA reactivity symptoms or heart rate and heart rate variability as these were distinct constructs with independent hypotheses.

Results

Affect.

Mother Affect. There were no overall group differences for mother NA reactivity across interactions ($\beta = -0.676$, $SE = 0.388$, $p = 0.082$, 95% CI [-1.436 – 0.084]), but as shown in Figure 1a. there was a significant group by task effect such that Depressed Mothers demonstrated greater reactivity of NA during the PSI task ($\beta = -1.500$, $SE = 0.511$, $p = 0.004$, 95% CI [-2.502 – -0.498]). In contrast, there were no overall group differences in PA reactivity across interactions ($\beta = -0.716$, $SE = 0.923$, $p = 0.439$, 95% CI [-2.526 – 1.094]) or group by task effect for PA reactivity ($\beta = 0.550$, $SE = 1.223$, $p = 0.653$, 95% CI [-0.091 – 0.120]).

Figure 1. Negative Affect Reactivity. A. Maternal Negative Affect Reactivity. B. Adolescent Negative Affect Reactivity. Note: Plots were made using code from Allen et al. (2019) and show raw data, density plots, boxplots, and standard error.

Adolescent Affect. There were no overall group differences for adolescent NA reactivity across interactions ($\beta = 0.247$, $SE = 0.193$, $p = 0.203$, 95% CI [-0.132 – 0.626]), but as shown in Figure 1b, there was a significant group by task effect such that High-Risk Adolescents demonstrated greater reactivity in reported NA during the PSI task ($\beta = -1.191$, $SE = 0.263$, $p < 0.001$, 95% CI [-1.708 – -0.675]). In contrast, there were no overall group differences in PA reactivity across interactions ($\beta = -0.425$, $SE = 0.598$, $p = 0.478$, 95% CI [-1.596 – 0.747]) or group by task effects for PA reactivity ($\beta = 0.643$, $SE = 0.825$, $p = 0.436$, 95% CI [-0.974 – 2.260]).

Physiology.

Mother Heart Rate. There were no overall group differences for mother heart rate reactivity across interactions ($\beta = 1.189$, $SE = 0.707$, $p = 0.093$, 95% CI [-0.197 – 2.574]) and no significant group by task effect ($\beta = -0.146$, $SE = 0.517$, $p = 0.777$, 95% CI [-1.160 – 0.868]).

Mother RMSSD. There were no overall group differences for mother RMSSD reactivity across interactions ($\beta = 0.552$, $SE = 1.825$, $p = 0.763$, 95% CI [-3.026 – 4.129]) and no significant group by task effect ($\beta = -0.756$, $SE = 1.354$, $p = 0.577$, 95% CI [-3.409 – 1.896]).

Mother PEP. There were no overall group differences for mother PEP reactivity across interactions ($\beta = 2.039$, $SE = 2.804$, $p = 0.468$, 95% CI [-3.456 – 7.533]) and no significant group by task effect ($\beta = -2.314$, $SE = 1.733$, $p = 0.184$, 95% CI [-5.712 – 1.084]).

Mother SCL. There was an overall group difference for mother SCL reactivity across both interactions (see Figure 2a), such that Depressed mothers exhibited less SCL reactivity across tasks ($\beta = 0.495$, $SE = 0.204$, $p = 0.016$, 95% CI [0.096 – 0.894]), but there were no significant group by task effects ($\beta = -0.012$, $SE = 0.120$, $p = 0.918$, 95% CI [-0.247 – 0.222]), controlling for multiple comparisons ($p = 0.032$).

Figure 2. SCL Reactivity. A. Maternal SCL Reactivity. B. Adolescent SCL Reactivity. Note: Plots were made using code from Allen et al. (2019) and show raw data, density plots, boxplots, and standard error. SCL = Skin Conductance Level.

Adolescent Heart Rate. There were no overall group differences for adolescent heart rate reactivity across interactions ($\beta = 0.252$, $SE = 0.833$, $p = 0.763$, 95% CI [-1.382 – 1.885]) and no significant group by task effect ($\beta = -0.633$, $SE = 0.524$, $p = 0.227$, 95% CI [-1.661 – 0.395]).

Adolescent RMSSD. There were no overall group differences for adolescent RMSSD reactivity across interactions ($\beta = -1.164$, $SE = 2.982$, $p = 0.697$, 95% CI [-7.008 – 4.681]) and no significant group by task effect ($\beta = 2.810$, $SE = 2.239$, $p = 0.211$, 95% CI [-1.579 – 7.198]).

Adolescent PEP. There were no overall group differences for adolescent PEP reactivity across interactions ($\beta = 1.892$, $SE = 1.803$, $p = 0.295$, 95% CI [-1.642 – 5.425]) and no significant group by task effect ($\beta = -0.001$, $SE = 0.752$, $p = 0.999$, 95% CI [-1.475 – 1.474]).

Adolescent SCL. There was an overall group difference for adolescent SCL reactivity across interactions (see Figure 2b), such that High-Risk adolescents exhibited less SCL reactivity across tasks ($\beta = 1.016$, $SE = 0.044$, $p = 0.001$, 95% CI [0.421 – 1.612]), but there was no significant group by task effects ($\beta = -0.230$, $SE = 0.195$, $p = 0.241$, 95% CI [-0.612 – 0.153]), controlling for multiple comparisons ($p = 0.002$).

Post-Hoc Analyses.

Because reactivity differences during dyadic interactions could be attributable to the behavior of the dyadic partner, given evidence that depressed persons (in this study and others) have more adverse family interactions, we examined post-hoc between group differences in outcome measures, controlling for interactants' behavior (adolescent behavior in maternal models and maternal behavior in adolescent models). These models can be found in supplemental materials. Findings remained consistent after controlling for interaction behaviors.

Discussion

The current study was designed to examine the association of maternal depression with both mother and adolescent offspring's self-reported affect and autonomic reactivity during positive and negative interaction tasks. The results provide partial support for the hypotheses. Consistent with hypotheses, both Depressed mothers and their High-Risk adolescents displayed significantly greater reactivity in self-reported NA during the negative interaction task. This finding is consistent with 1) research showing that depression is associated with greater NA reactivity to negative life circumstances (Bylsma et al., 2011; Myin-Germeys et al., 2003; van

der Stouwe et al., 2019; van Winkel et al., 2015), 2) prior research showing that depressed persons exhibit greater reactivity to emotional stimuli that is social in nature and indicative of threat (N. B. Allen & Badcock, 2003; Price et al., 1994), 3) prior research that showing that during negative interactions mothers and their depressed adolescents exhibit greater levels of NA behavior (Sheeber et al., 2012), 4) females with depressed mothers exhibit greater NA reactivity to stress than females without depressed mothers (Waugh et al., 2012), and 5) the theory of stress sensitivity by which the tendency to react stronger to stress has been shown to predict increased depressive symptomatology and development of depression (Wichers et al., 2007, 2009). Importantly, these findings contrast with prior research showing that those with depression exhibit blunted affective reactivity in laboratory studies using less personally relevant stimuli (Bylsma et al., 2008). These findings also elucidate that NA reactivity may be context dependent, such that depressed mothers and their offspring exhibit typical levels of NA reactivity during positive interactions, with differences from non-depressed mothers and Lower-Risk peers only arising in more challenging situations. The finding regarding adolescents is even more compelling when noting that the model controlled for adolescents' own mental health symptoms and post-hoc analyses also controlled for maternal behaviors during the interaction -- indicating that it was maternal depression, rather than adolescents' own mental health or maternal behaviors that related to increased adolescent NA reactivity.

Also in line with our hypotheses, Depressed Mothers showed blunted sympathetic (i.e., SCL) reactivity across tasks when compared to Non-Depressed Mothers even after controlling for covariates (medication use, age, and adolescent behaviors in post hoc analyses) and multiple comparisons. These findings are consistent with research indicating that those with depression and those with increasing depressive symptoms have blunted autonomic reactivity (Carroll et al.,

2007; Phillips, 2011; Salomon et al., 2009; Schiweck, Piette, Berckmans, Claes, & Vrieze, 2019; York et al., 2007), particularly during interpersonal interactions that elicit a beta-adrenergic response (Salomon et al., 2009; Salomon et al., 2013). This blunted or biological disengagement of sympathetic activity is in line with decreased motivation in those with depression (Salomon, Bylsma, White, Panaite, & Rottenberg, 2013).

In contrast to our hypotheses, which were based on the theoretical assumption that maternal depression may act as a form of social threat for adolescents that results in increased physiological reactivity, our findings indicated that High-Risk Adolescents exhibited the same blunted sympathetic reactivity via SCL across both positive and negative interaction tasks as displayed by their Depressed Mothers. This result is therefore more consistent with the alternative theoretical approach which would predict that adolescents of depressed mothers would exhibit a more blunted autonomic reactivity profile associated with depression (Salomon et al., 2013). These findings extend prior literature by 1) replicating findings of blunted autonomic reactivity in those with depression, 2) extending these findings to the adolescent offspring of depressed mothers, and 3) elucidating that this process may be context independent, in that it was consistent across tasks with varying emotional demands. Though of course the contexts were similar in being parent-child interactions within a laboratory setting, leaving open the question of how other contexts could influence the findings. Again, the finding among adolescents is even more compelling when noting that the model controlled for adolescent's own mental health symptoms, medication use, age, gender, and in post-hoc models maternal behaviors during the interaction tasks -- indicating that it was maternal depression, rather than adolescent's own mental health or maternal behaviors that related to sympathetic reactivity. Furthermore, this blunted sympathetic process exhibited by adolescents of depressed mothers

may indicate withdrawal and disengagement during the interaction tasks, which would be consistent with a pattern of learned helplessness during which there is a blunting of acute stress reactivity across time (Maier & Seligman, 2016).

Finally, there were no differences in either PA reactivity nor any measure of autonomic reactivity, except for skin conductance, between High-Risk and Lower-Risk adolescents and Depressed and Non-Depressed mothers, which may reflect that the interaction tasks didn't elicit the type of reactivity we were measuring, such that the task had no association with PA in adolescents or mothers ($p > 0.99$) in post-hoc analyses. Perhaps an interaction task that involved more high arousal positive affect, such as those displayed more commonly among friends (Dahl et al., 2018) than between parents and adolescents would have elicited a difference (Dahl et al., 2018).

Limitations and Future Directions.

While the present study had significant strengths such as using a multimethod assessment of reactivity across affective self-report and autonomic psychophysiology, there were a number of limitations that should be noted. First, this was a cross-sectional study, which precluded the ability to study developmental timing effects within adolescence (i.e., early, middle, late adolescence); future studies would benefit from longitudinal designs to extend these findings. Second, although the EPI and PSI tasks significantly discriminate between groups in affective behavior, tonic levels of psychophysiology (Nelson et al., Preprint) and some indices of reactivity, the tasks capture a relatively narrow behavioral context. As we have argued previously, future studies might address this limitation by utilizing passive sensing technologies (i.e., wearable, smartphone, and smart home devices) to increase the range of environmental contexts examined (Allen, Nelson, Brent, & Auerbach, 2019; Nelson & Allen, 2018, 2019) to

bring this type of research out of the lab and into real world contexts. Third and relatedly, assessing stress reactivity on one day does not provide the level of temporal detail required to generalize one stress response to repeated and chronic stress of daily life. Therefore, there is a need to address the gap between an acute stress response and chronic stress (Rohleder, 2019). Again, utilizing passive sensing technologies may allow for the unobtrusive and temporally fine-grained data collection that could more accurately characterize stress response within and across contexts and time (Nelson & Allen, 2019).

Conclusion.

This study was the first to examine the role maternal depression plays in both maternal and adolescent affect and autonomic reactivity during positive and negative interaction tasks. Overall, our findings indicate that both depressed mothers and their adolescent offspring exhibit similarly exaggerated NA reactivity, specifically during negative interactions, while they also show blunted sympathetic activity across positive and negative interactions. These findings indicate that dysregulated NA during negative interpersonal interactions and blunted sympathetic activity across positive and negative interactions may be two mechanisms that are influenced by depression within and across generations.

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